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The effects of progressive anemia on jejunal mucosal and serosal tissue oxygenation in pigs.

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Anemia may promote intestinal hypoxia. We studied the effects of progressive isovolemic hemodilution on jejunal mucosal (P_{O_2muc}), and serosal tissue oxygen tension (P_{O_2ser} , Clark-type surface electrodes), mucosal microvascular hemoglobin oxygen saturation (HbO_2muc), and hematocrit ($Hctmuc$; tissue reflectance spectrophotometry) in a jejunal segment. Twelve domestic pigs were anesthetized, paralyzed, and mechanically ventilated. Laparotomy was performed, arterial supply of a jejunal segment isolated, and constant pressure pump perfused. Seven animals were progressively hemodiluted to systemic hematocrits (Hct_{sys}) of 20%, 15%, 10%, and 6%. Baseline for P_{O_2muc} , P_{O_2ser} and HbO_2muc was 23.5 ± 2.1 mm Hg, 57.5 ± 4 mm Hg, and $47.0\% \pm 6.4\%$ which were not different from the five controls. Despite a significant increase in jejunal blood flow, jejunal oxygen delivery decreased and oxygen extraction ratio increased significantly at Hct_{sys} 10% and 6%. P_{O_2ser} decreased significantly below or at Hct_{sys} of 15%, whereas P_{O_2muc} and HbO_2muc were maintained to Hct_{sys} of 10%, but less than 10% HbO_2muc and mesenteric venous pH decreased significantly, implying that physiological limits of jejunal microvascular adaptation to severe anemia were reached. Decrease of $Hctmuc$ was less pronounced than Hct_{sys} . In conclusion, redistribution of jejunal blood flow and an increase in the ratio of mucosal to systemic hematocrit are the main mechanisms maintaining mucosal oxygen supply during progressive anemia.

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